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An extensive review has been completed of the hormonal regulation of fluid and electrolyte balance during sedentary exposure to or exercise in the heat. The review focuses mainly on human responses although examples from animal studies are also included. The effects of exogenously administered hormones has been discussed followed by sections on hormonal response to sedentary heat exposure and exercise in the heat. Consideration of the exacerbative effects of hypohydration on the hormonal responses is followed by a section on electrolyte supplementation and the effects of heat acclimation. The review concludes with a discussion of the role of plasma volume in modifying the endocrinological responses as well as suggestions for future studies in this area.

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HORMONAL REGULATION OF FLUID AND ELECTROLYTES: EFFECTS OF HEAT EXPOSURE AND EXERCISE IN THE HEAT

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INTRODUCTION

A review of the endocrine factors which subserve fluid and electrolyte regulation during acute or chronic sedentary heat exposure or during exercise in the heat necessarily focuses on two target organs - the sweat gland and the kidney. While the acquisition of heat acclimation in humans is partially characterized by a striking ability to secrete increased quantities of a more dilute sweat (3,121,131), there are relatively few studies in the scientific literature describing the direct hormonal control of sweat secretory rate, composition, or total output. Further, studies which have reported these direct effects of exogenously administered hormones (e.g. aldosterone, angiotensin I or II, antidiuretic hormone or vasopressin) on sweat gland activity provided, at best, inconsistent results with various investigators reporting increased, decreased, or no effects on sweat secretion.

Effects of Exogenous Hormones

In an attempt to confirm the responsivity of the eccrine sweat gland to antidiuretic hormone (vasopressin, VP) stimulation, Fasciolo et al. (48) injected VP subdermally at 3 sites (forearm, abdomen, thigh) in 6 male volunteers and collected sweat samples for three 40 min periods at each site and the contralateral control area. Sweating was induced by heat exposure or Mecholyl administration, and the results indicated that, indeed, sweat secretion was generally reduced by VP treatment; moreover, the sodium (Na^+) concentration of the sweat was increased, and these investigators concluded that VP had a direct effect on sweat duct permeability and water reabsorption.

In a very early experiment Ladell (109) injected deoxycorticosterone acetate intramuscularly in acclimated subjects and collected forearm sweat during work in a hot environment. The results indicated that the hormone, acting directly on sweat gland secretion, elicited a 30% reduction in the sodium chloride (NaCl) content of the sweat. At approximately the same time Conn et al. (29) also administered deoxycorticosterone acetate to men before, during, and after acclimation to heat, and reported that the concentration of NaCl lost in sweat was significantly lowered and that cessation of hormonal administration promptly resulted in increased salt losses even in acclimated subjects. In fact, they concluded that the exogenous administration of hormone repressed endogenous mineralocorticoid secretion, and full recovery from this hyposecretory activity required several days.

Twenty years later Collins (24) administered d-aldosterone at 6 h intervals and induced sweating by intradermal injection of methacholine. He reported that between 12-30 h following the first aldosterone injection, the

sodium/potassium ratio was maximally depressed, urine flow and sweat secretion were both attenuated, and 2 days were necessary for these variables to return to normal levels after the final injection of aldosterone. These studies led Braun et al. (18) to administer d-aldosterone exogenously to young, healthy volunteers in an attempt to accelerate the acquisition of heat acclimation. They concluded (18) that while the total time necessary for full acclimation was not reduced, performance time was increased while mean rectal temperature and heart rate were reduced during the first several days of exercise in the heat.

In other related studies Gibinski et al. (69) administered 1.25 mg aldosterone acetate or 0.5 and 1.0 g Aldactone (spironolactone) before exposure to a hot, wet ($48^{o}/40^{o}C$, db/wb) environment and measured several sweat variables during the heat exposure. They (69) observed no effects of either intervention on sweat chloride, sodium, and potassium (K). While urinary Na/K ratios were decreased by heat exposure and heat exposure plus aldosterone administration, the antagonistic effects of Aldactone on aldosterone prevented Na conservation and urinary Na/K ratios were unaffected (70) in this trial. Since Aldactone affected urinary sodium conservation while sweat sodium concentrations were unaffected by this treatment, these workers concluded that the mechanisms of action in the sweat glands and kidneys "differ entirely".

When Zgoda et al. (168) exposed 10 test subjects to $49^{o}C$ for 2 h/d for 5 d and administered VP intranasally to 5 subjects daily, they reported that the VP-treated group manifested decreased rectal temperature (Tre) and heart rate during the heat exposure. In a related paper (11) a 10 min work interval was added at the conclusion of the 2 h heat exposure, and they observed no effects on sweat rate while "psychophysiological" improvement was noted in subjects receiving the peptide. Senay and van Beaumont (153) injected vasopressin (Pitressin®) intramuscularly before a final hour of heat exposure with water replenishment and observed no effects of VP administration on evaporative water loss while urinary water loss was reduced in both subjects. Ratner and Dobson (127) had earlier reported that VP administration and heat exposure elicited a marked increase in urinary osmolality with no changes in sweat osmolality. In a more recent paper Gibinski et al. (71) also administered VP to a group of volunteers and observed no effects on sweat rate, osmolality, and electrolytes. In the latter experiments (71) no effects on urine osmolality were reported. Thus, despite a significant number of investigations, uncertainties persist with respect to the effects of exogenously administered hormones on the volume and chemical composition of both sweat and urine.

Clearly, these sometimes divergent observations, especially with respect to exogenous VP, underscore the fact that the control of fluid and electrolyte balance in higher organisms is an extremely complex and exquisitely controlled system involving certainly not only hormonal, but also neural and other physiological factors (106). This topic has been the subject of frequent and extensive reivews and is well beyond the scope and intent of the current paper (For general reviews see: 9,13,81,97,128). In addition, there are several earlier reviews available which readers might wish to consult concerning water and electrolyte balance and metabolism during sedentary or active exposure to hot environments (23,28,46,80,112,166). Although Doucet (40) has recently noted that there are over 20 hormones controlling kidney function and regulation, most of these have not been investigated for their potential impact in controlling fluid and electrolyte balance during heat exposure.

Generally, it has been concluded that sedentary exposure to or exercise in a hot environment for at least 1 h by non-acclimated individuals without fluid replenishment is usually accompanied by a reduction of the interstitial and plasma volumes (56) and a significant loss of Na^{+} in sweat (100). Additionally, blood flow is redistributed from the visceral circulation

(135,136) to the exercising muscles or skin surface for heat dissipation. The
early decrements in plasma volume (83) and renal blood flow (143) stimulate
the activity of the renin-angiotensin system (47,52) as well as the synthesis
and release of aldosterone (34,37) (Fig.1). If a heat/exercise regimen is
maintained for at least several consecutive days, stimulated aldosterone (A)
and vasopressin (VP) secretion will effect precipitous declines in urinary
volumes (23,46) in euhydrated and, especially, hypohydrated individuals and
Na^+ excretion in the urine (62) followed soon thereafter by significant
decrements in sweat Na^+ (12). Because of the marked reductions in sweat and
urinary Na^+ loss, Na^+ balance may be rapidly re-established even in the
presence of only moderate levels of Na^+ consumption, and this may contribute
to the beginnings of plasma volume (PV) expansion associated with heat
acclimation.

If daily exercise in a hot environment persists, water and electrolyte
equilibrium is usually fully re-established after approximately 1 week of the
exercise/heat regimen (6) as long as fluid and electrolyte replacement is
adequate. Certainly by this time sweat and urinary Na^+ losses have been
minimized even while sweat volume has been significantly increased (160),
urinary output is adequate but attenuated, and resting PV may be expanded over
a broadly reported range, e.g. 7% (15), 15% (85), and 23% (147) while total
body water may be expanded by approximately 6% (32). The magnitude of the
hormonal responses and adjustments which help to maintain fluid and
electrolyte balance during acute or recurrent sedentary heat exposure or
exercise in the heat may be affected by the acclimation (or training) status
of the individual, the hydrational status, the nutritional status (expecially
with respect to electrolytes), the duration and intensity of the stress, as
well as additional variables which may be introduced by the actual
experimental scenario. In this chapter we will attempt to consider each of
the important factors relevant to the hormonal control of or the hormonal
response to electrolyte and fluid perturbations during sedentary or active
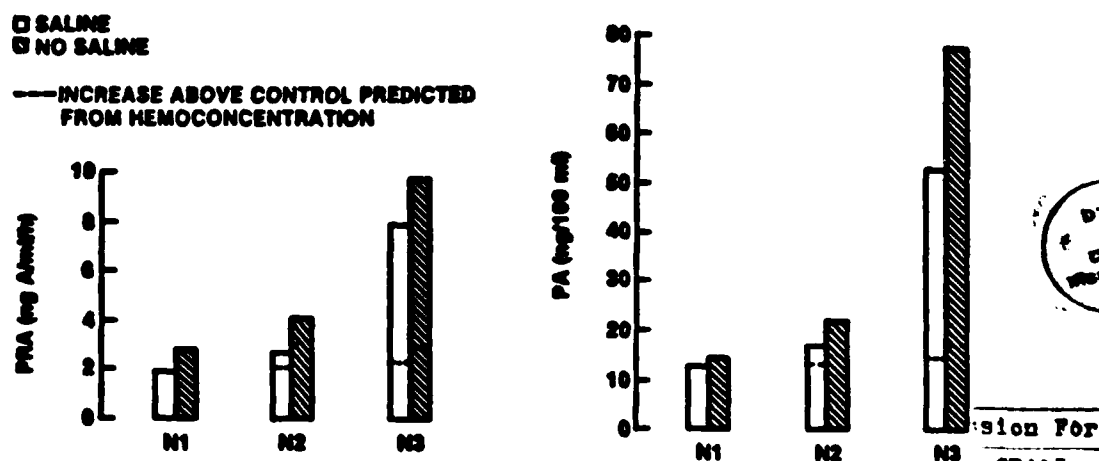exposure to heat stress.

Fig. 1 Effects of consumption of 0.75 liters of 1% saline on plasma renin
activity and plasma aldosterone levels during a heat acclimation regimen. The
N1 sample was taken after 25 min. rest at 40°C, the second (N2) sample 5 min
after 30 min intermittent exposure to a hot bath (40°C), and the final sample
(N3) during the final minute of 10 min continuous exercise on a bicycle
ergometer (40°C, 75W). The rest interval, hot bath, and exercise period were
contiguous. The blood samples were taken on alternate days during an 11 d
heat acclimation program. Mean values for 6 young adult males are depicted.
Results of this study emphasize the importance of exercise (N3), with its
concurrent diversion of blood flow from the viscera, in stimulating the
biosynthesis and reducing the clearance of PRA and PA. (Redrawn from: Davies
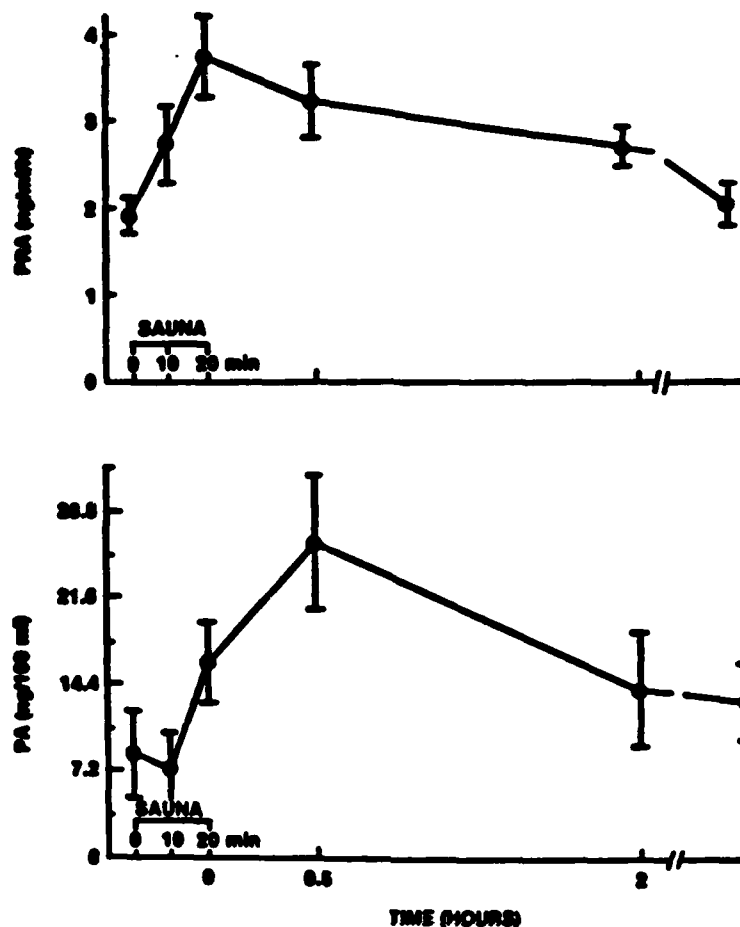et al., J. Appl. Physiol., 50, 605, 1981 with permission of the publisher)

Fig. 2 Effects of intense heat (85-90°C) on plasma renin activity and
aldosterone levels in 6 healthy adult males who were experienced sauna users.
Mean values + SEM are depicted. Venous blood samples were taken at the times
noted while subjects remained sedentary. The rapidity of the PRA response may
indicate the importance of diversion of blood flow from the renal bed in
increasing PRA; the greater (4X) response of PA may implicate the generalized
adrenocorticotrophic effect. (Redrawn from: Kosunen et al., J. Appl.
Physiol., 41, 323, 1976 with permission of the publisher)

SEDENTARY HEAT EXPOSURE

Perhaps the most rapid sedentary exposure to heat stress eliciting
significant alterations in hormones controlling plasma volume was reported by
a Helsinki group using conditions "typical of a Finnish sauna bath". Kosunen
et al. (108) and Adlerkreutz et al. (2) reported the hormonal responses of
experienced sauna users to 20 min exposure at 85-90°C. Their results
indicated that just 10 min after exposure to the intense heat stress, levels
of plasma renin activity (PRA) and angiotensin II were significantly
increased; increments in plasma aldosterone (PA) levels were delayed until 20
min after exposure with even greater elevations observed 0.5 h after
completion of the heat stress. Generally, hormonal levels had returned to
control ranges 6 h following heat exposure (Fig. 2).

These extremely rapid hormonal responses suggest that fluid shifts and
shifts in blood flow-cardiac output distribution are stimulatory to
endocrinological responses in the absence of notable fluid loss. Rowell (134)
has estimated that skin blood flow can be increased to 7-8 L/min during severe
heat stress, and concluded that splanchnic vasoconstriction was initiated
primarily by the sympathetic nervous system since increments in PRA occur
concomitantly with elevations in heart rate and plasma norepinephrine levels.
Baroreceptors may be initially involved followed by the activation of volume,
osmo-, and thermoreceptors as the stress persists eliciting plasma volume
decrements, osmolality increments, and elevations in core temperature.

Dumoulin et al. (41) used an 80°C environment and 20 min exposure time to effect significant elevations in both PRA and PA. In their earlier report Kosunen et al.(108) indicated that no changes occurred in PV as estimated from hematocrit, hemoglobin, and plasma protein (PP) values. The rapidity of the hormonal changes in these studies is noteworthy; just 10 min after intense heat exposure, circulating indices of fluid and electrolyte conservation and splanchnic and renal vasoconstriction were evident. The spontaneity of these responses suggests that increased circulating hormonal levels can be elicited by reduced hepatic and renal clearance in addition to accelerated biosynthesis of these hormones. Collins et al. (26) have reported that the metabolic clearance rate of aldosterone was reduced from 77 L/h to 57 L/h when deep body temperature was raised by 1.08°C. The effects of acclimation on the clearance of fluid and electrolyte regulatory hormones have not been assessed (25).

Rocker et al. (132) extended the heat exposure (70-75°C) to 4 h with a 10 min interval at 19-21°C during each hour. With no fluid replenishment this test scenario effected a dehydration of 2.4 kg or 3.4% of pre-exposure body weight. In addition to observing a 100% elevation of plasma vasopressin (PVP) at the end of the heat exposure, which was further increased to approximately 160% 90 min later, they also reported significant increments in plasma osmolality and concomitant decrements in plasma volume, thus emphasizing the importance of the duration of the stress in affecting physiological responses and the impact of fluid loss in addition to fluid shifts in eliciting marked hormonal elevations. Earlier, these workers had employed the same experimental protocol (133) to show that untrained women manifested smaller reductions in plasma volume than men.

While sedentary exposure to sauna temperatures thus elicited rapid and intense alterations in circulating hormone levels when large shifts in fluid volumes and blood flow distribution were occurring, much of the experimental work in humans as well as higher animals has been accomplished under much lower ambient conditions. Szczepanska-Sadowska (161) used pre-implanted thermodes near the pre-optic area of dog forebrain to raise brain temperature by 1.5°C in the absence of generalized heat stress and remarkably observed 10-fold elevations in circulating VP levels just 10 min after heating. Eisman and Rowell (43) exposed 6 male baboons to environmental temperatures ranging from 42-49°C and observed 24% reductions in renal blood flow per °C rise in core temperature giving rise to 98% increments in PRA per °C elevation. This same group of investigators would later (47) use human subjects in water-perfused suits to show that raising Tre by 1°C elicited increases in PRA of 135% and in splanchnic vascular resistance of 73%. B-blockade, induced by propranolol administration, reduced PRA, but elevations in splanchnic vascular resistance were less affected. These investigators speculated (47) that while PRA is effective in eliciting visceral vasoconstriction when heat stress is mild, increased stress intensity may stimulate sympathetic nervous system-induced vasoconstriction. Rowell (134) later hypothesized that angiotensin II may have more marked effects on the renal vs the splanchnic circulation.

Thus, in the absence of exercise, exposure to heat stress is accompanied by reduced urinary fluid and electrolyte loss partially under the influence of increased PRA (43,47,49) and aldosterone (49,55). Simultaneously, cutaneous vasodilation, occurring in humans generally over the entire surface of the body from the lower extremities to the cheek and ear region (148), permits the increased peripheral blood flow provided by the enormous increments in cardiac output which results in similarly increased evaporative, radiative, and convective heat loss under appropriate conditions. Of course, in the passive mode the absence of increased metabolic heat production from muscular activity results in comparably lower sweat rates to maintain thermal equilibrium under specified conditions of ambient temperature and duration of exposure.

Elizondo et al. (44) used an environmental temperature of 36-40°C to induce sweat rates of up to 0.65 mg/cm²/min, and reported that while sodium

concentration (Na) increased with increasing sweat rate, the potassium concentration (K) of the sweat was reduced. These investigators (44) used local skin heating (to 42°C) and arterial occlusion to increase or decrease, respectively, sweat output, but did not speculate on the control of electrolyte loss in the sweat. Harrison (82) investigated the effects of 2 h sedentary exposure to 48°C, and observed that while plasma Na^+ and K^+ were unaffected, there occurred a linear decrease of water from the intravascular space which could be correlated with either increasing hematocrit or protein concentration. Additionally, van Beaumont et al. (164) used similar conditions (sedentary, 45°C, 3 h) to demonstrate that thermal dehydration (1.9% loss in body weight, bw) was accompanied by proportionately greater reductions in plasma volume (5.9%) than heat stress alone. These investigators (164) concluded that the dynamics of hemodilution and hemoconcentration during sedentary heat exposure may be related to the onset of sweating, initial sweat rate, and total sweat loss.

To this point, the results from studies using initially euhydrated human subjects in climatic chambers, sedentary exposure to heat ranging from moderate to intense, and durations ranging from a fraction of an hour to several hours indicated that hormonal adjustments conducive to fluid and electrolyte conservation are generally rapid and consistent. Surprisingly, Candas et al. (20) showed that marked hidromeiosis (reduction in sweat rate due to skin wettedness with prolonged sweating) during repeated exposures to hot, humid conditions did not elicit any changes in PA, PRA, or PVP. During active heat dissipation in the passive mode, visceral blood flow is attenuated and the peripheral share of cardiac output is increased to as much as 60% of the total (134). Despite the hormonal adjustments designed to conserve fluid and electrolytes, dehydration may occur and plasma volume may be noticeably reduced if fluid replenishment is inadequate or heat stress is intense and prolonged.

EXERCISE IN THE HEAT

When physical exercise is added to the stress of the hot environment, then the cascade of endocrinological and physiological adjustments necessary to meet metabolic and thermoregulatory demands is increased. When we exercised rats in the heat (35°C) and removed a blood sample for analysis when Tre reached 40°C, we observed significant elevations in PA in this sample which was taken just 8.25 min (mean) after the initiation of exercise (61). In humans increased cardiac output and blood flow to sustain the metabolic demands of the working muscles must be partially distributed also to the skin surface for sweat secretion and evaporation; the percentage of cardiac output to each may be determined by the work intensity and the ambient temperature. In any event, the surfeit of deep body heat, which can raise core temperatures to injurious levels, must be transferred to the periphery for radiative, convective, or evaporative dissipation. To meet the enormous demands placed upon the cardiovascular system during exercise in the heat (120) in man or animals, plasma volume must be protected and sustained. Fortunately, healthy humans ordinarily possess physiological and endocrinological mechanisms to defend plasma volume, reduce electrolyte loss, promote heat loss, and attenuate physiological strain during work in the heat.

Even at apparently moderate, but unspecified, environmental temperatures, Convertino et al. (30) observed that increasing the workload (bicycle-ergometer, 100, 175, 225 watt, W) among 15 healthy male test subjects elicited linear (with workload) increments in PRA which were significant at all three work intensities. Circulating VP, however, manifested a more curvilineur increase which was markedly elevated at the highest work load. Decrements in plasma volume were also linearly (inversely) correlated with workload (-3.7% at 100 to -12.4% at 225 W). While changes in circulating Na^+ concentration and osmolality were closely correlated with changes in VP, these variables were not correlated with PRA. These results indicate that PRA responds

linearly to the reduction in renal blood flow due to the diversion of cardiac output to the exercising muscle mass. However, PVP release may be more closely correlated with elevated osmolality and plasma sodium levels. When the same group of investigators (31) later studied the effects of training on the responses of many of these same variables at an ambient temperature of 25°C, they reported that training increased resting PV by 12.3% although resting baseline levels of PRA and PVP were unaffected by training. Further, they demonstrated that the increments in PRA and VP during exercise observed prior to training were attenuated by the training regimen. The effects of training on plasma protein concentrations and total circulating protein were not evaluated. It is noteworthy that, subsequent to training, the percentage decreases in plasma volume were reduced following 175 W and 225 W exercise (31) since total PV was increased by the training regimen. Also, because of the increased VO_2 max (11.2%), each workload represented a lowered relative intensity (% VO_2 max) and therefore a decreased physiological strain on the test subjects. This is certainly suggestive evidence that the magnitude of the response of the fluid regulatory hormones may be affected by the relative exercise intensity and the plasma volume itself.

Interestingly, when Finberg et al. (52) compared the responses of adult males to walking in the heat (level treadmill, 1.3 m/s, 90 min, 50°C) during the summer and winter months, they observed again an attenuated response of PRA during the summer trial which may be related to the increased plasma volume of natural acclimatization during the summer months. They also reported (52) that replacement of the estimated fluid loss during the 90 min walk with water and NaCl reduced the intensity of the PRA response pattern in 4 out of 5 subjects (Fig. 3), thus indicating probably that fluid replenishment helped to sustain renal blood flow, albeit at reduced levels. When Costill et al. (33,34) used exercise (60 min, 60% VO_2 max) at 30°C to study urinary sodium loss, they observed that levels of PRA and PA were elevated during and immediately following exercise in the heat, and returned to control levels usually by 6-12 h following exercise completion. Additionally, they reported that this scenario consisting of a single exercise interval was effective in reducing urinary Na^+ and Cl^- concentrations for up to 48h, presumably due to the effects of the single elevation in PA levels subsequent to exercise.
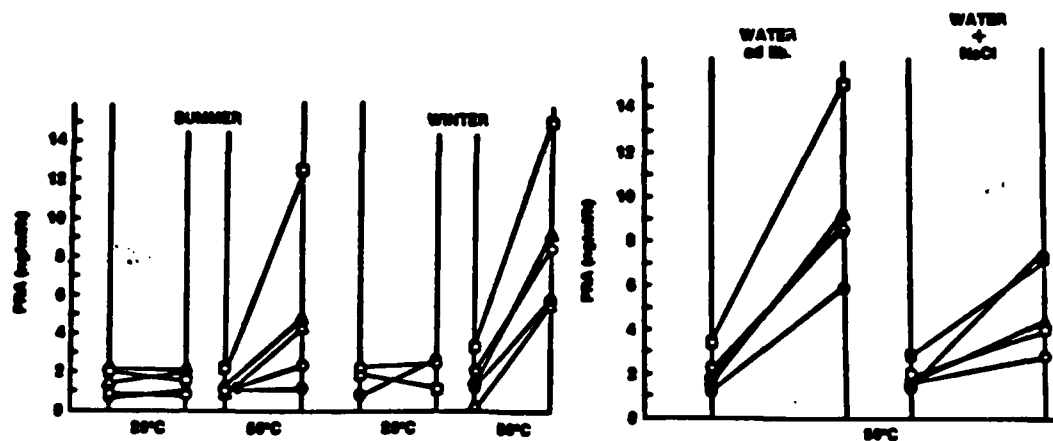

Fig. 3 Effects of natural acclimatization and salt replacement on the responses of PRA to treadmill exercise at 25°C and 50°C in healthy, male volunteers. Individual data are depicted. In each section pre-values (on the left) are connected to post-values (on the right). Summer trials were conducted in September and October while winter tests were executed in December and January. Salt ingestion was based on the estimate that the NaCl content of sweat lost was 400 mg/100 ml, and replaced at 15 min intervals during the 90 min walk at 4.7 km/h. The increased intravascular volume of summer may blunt the PRA response in comparison to the winter trial as the NaCl ingestion relieves the demand to replace the NaCl lost in sweat. (Redrawn from: Finberg et al., J. Appl. Physiol., 36, 519, 1974 with permission of the publisher)

Similarly, when Orenstein et al. (122) exercised normal young adult test subjects at 50% VO_2 max at 37°C, also for 90 min, they observed significant elevations in PRA and PA immediately subsequent to exercise, and these increments contributed to marked reductions in urinary Na^+ loss with no effects on serum Na^+. Alternatively, in these studies (122) cystic fibrosis patients manifested normal hormonal responses to heat/exercise stress and similar reductions in urinary Na^+, but serum Na^+ levels were also reduced, probably due to excessive loss in sweat. Interestingly, the cystic fibrosis patients, who can and do manifest the physiological advantages of heat acclimation (123) yet respond to heat stress with an increased loss of sweat Na^+ and Cl^- (122), thus demonstate normal responses to exercise in the heat with respect to PRA and PA.

Therefore, during exercise in the heat hormonal responses directed at fluid and electrolyte conservation are rapid and intense with probable persistent effects, and may be partially responsible for the defense of body water and extracellular fluid which help to maintain the efficiency of the cardiovascular and thermoregulatory systems. In one of the very few studies which has quantitated fluid- and electrolyte-regulatory hormone levels after heat injury, Aarseth et al. (1) observed significant elevations in PRA and PVP at rest following incurrence of the injury in 6 athletes who suffered heatstroke with Tre in excess of 42.0°C.

When humans exercise in the heat even for relatively brief intervals, thermal sweating without fluid replacement is accompanied by general decrements in body weight with proportionately more intense reductions in plasma volume. Diaz et al. (38) investigated the effects of posture on plasma volume changes during rest (45 min) and exercise (45 min) in a hot environment. Subjects worked on a bicycle ergometer (30% and 45% VO max, 49.5°C), in the upright, low-sit and supine postures without fluid replacement. During the total heat exposure, plasma volume was reduced by 20% in the upright, 16.1% in the low sit, and 13.3% in the supine positions with no differences noted between work loads. Body weight was reduced by only 1.3-1.4% in these experiments, thus eliciting substantial ratios in the percent decrease in PV versus percent decrease in body weight. No effects of posture were noted on either plasma protein levels or osmolality suggesting an isooncotic and isoosmotic fluid shift out of the plasma space, and, unfortunately, hormonal responses were not assessed.

When Harrison et al. (86) exercised adult unacclimated males for 50 min at 42°C and 3 months later at 30°C using the same experimental protocol without fluid replacement, they observed decrements in plasma volume of approximately 10-12% by the end of the exercise period in the fluid deprivation experiment. However, during a sedentary 50 min recovery period PV recovered to control or near control levels (depending on the method of calculation). These investigators (86) concluded that, as proposed by Senay (144, 145), the return of protein to the intravascular space via the lymphatic system during exercise and probably also during recovery contributed to an elevated oncotic pressure. This osmotic force in turn promotes water influx into the intravascular space at the expense of other fluid compartments. The same group (83) used radio-iodinated serum albumin to confirm that, following exercise, protein was transferred to the intravascular space more rapidly than it was lost through capillary permeability.

Senay and Fortney (149) compared the responses of untrained females to exercise (ergometer, 30% VO_2 max) at 16-20°C and 45°C and observed a PV decrement of nearly 13% in the thermoneutral environment and nearly 18% in the heat. While plasma protein increased markedly during the thermoneutral trial, no such increment was observed during the heat trial. This led Senay and Fortney (149) to conclude that the increased perfusion and, perhaps, permeability of the cutaneous capillaries, far more extensively perfused during exercise in the heat, counteract the retention or accumulation of

intravascular protein during the heat/exercise regimen. When Greenleaf et al. (76) used 45% VO_2 max and 40°C Ta as an exercise/heat stress, they observed decrements in PV of approximately 11% after 60 min. Thus, it is clear that, despite the hormonal responses which favor fluid and electrolyte retention, plasma volume is nonetheless decreased during acute heat/cycle exercise stress. When such loss of body water results in frank hypohydration, then the physiological and endocrinological adaptations conserving fluid and electrolytes become even more crucial to the prevention of heat/exercise injury (1) and maintenance of physical performance (5).

The results indicate that passive heat exposure, exercise, and exercise in the heat are all rapidly and variably stimulatory to increased circulating levels of PRA, PA, and PVP. PRA may be stimulated most effectively by a reduction in renal blood flow secondary to increased muscle or skin blood flow. PA apparently may be initially elevated by generally increased adrenocorticotrophic activity secondary to exercise/heat stress while PVP, initially elevated by acute heat exposure or exercise heat stress, may be more severely affected by increased plasma Na^+, osmolality, or decreased plasma volume secondary to hypohydration.

HYPOHYDRATION

For those readers interested in the comparison among species, the apparent universality of the endocrinological responses to heat/hypohydration has been confirmed in a wide range of experimental animals. For example, Keil and Severs (98) observed in rats that 48 h of water deprivation was effective in eliciting marked elevations in plasma vasopressin levels, but these increments were attenuated with increasing weight of the experimental animals. Further, they reported (98) that the imposition of a non-thermal secondary stress (ether, acceleration) tended to reduce the elevations in PVP effected by hypohydration. Kenyon et al. (99) observed that when 1 ug/d of aldosterone was administered to adrenalectomized rats, the animals produced a small volume of more concentrated urine and plasma K^+ levels were increased. When rats were passively exposed to an ambient temperature of 40°C, PVP levels were increased and the increments were surprisingly exacerbated by propranolol treatment (77). Arad et al. (4) examined the hormonal, fluid, and electrolyte responses in domestic fowl and noted that hypohydration (13% bw, 48 h water deprivation) increased PA, PVP, Na^+, and osmolality, all of which were exaggerated when hypohydration was combined with heat exposure (final ambient = 42°C) and attenuated during a rehydration interval (30 min); however, heat exposure alone had no effects on these variables. During hypohydration and heat exposure, core temperature increased to 43.7°C from 42.8°C (heat alone); similarly, plasma osmolality increased from 319 to nearly 360 mosm/kg, respectively.

Interestingly, Thrasher et al. (163) reported in dogs that 24 h of water deprivation (~ 5% body weight loss) caused significant elevations in Na^+, osmolality, PVP, and PRA which had returned to control levels after 1 d of rehydration. Alternatively, PA was not significantly affected during dehydration, but peaked at 24 h of recovery. They (163) attributed this 24 h peak to a decrement in plasma Na^+ occurring upon rehydration which was countermanded by this PA response, and hypothesized that a natriuresis during hypohydration helped to reduce the magnitude of the increments in plasma osmolality. However, using hyperhydrated goats Augustinsson et al. (8) reported no increments in excretion of VP and a decrease in PA during acute heat exposure (45°C); they attributed these observations to the significant respiratory alkalosis which developed by 60 min in this species.

Finally, even in animals as large as domestic cattle, El-Nouty et al. (45) reported that acute (8 h) heat stress (35°C) elicited a mean elevation in Tre of 2°C and a significant elevation in PVP which was further increased when water was withheld for 30 h. During 24 h of heat exposure PA levels were

significantly depressed in these experiments (45), and recovered toward control levels during dehydration. Again, they attributed the decrement in PA to the increased plasma Na$^+$ observed during heat stress. Thus, although the responsivity of the plasma hormones to hypohydration has been validated in a wide range of experimental species, the vast majority of this research has been accomplished using human test subjects.

In humans the effects of hypohydration during heat exposure and exercise in the heat have been extensively documented. Even in well heat-acclimated subjects, Strydom and Holdsworth (159) observed that during work in the heat increasing severity of hypohydration (3-5% and 5-8% body weight decrements) caused exaggerated increases in rectal temperature and heart rates while sweat rates were reduced. In these studies optimal performance (i.e. minimal physiological strain) was observed when test subjects were compelled to rehydrate at 15 min intervals; it is generally believed that complete rehydration, even when palatable and potable fluids are conveniently available and drinking is encouraged, is ordinarily delayed during and immediately after exercise in the heat - a phenonemon termed voluntary dehydration (74).

The increased physiological strain of hypohydration partially results from the significant decrements in PV and increases in osmolality which usually occur concomitantly with hypohydration, resulting in decreased sweat rates and reduced skin blood flow (138). For example, Saltin (138) observed that hypohydration, elicited by thermal exposure to a 5.2% reduction in body weight, may be accompanied by reductions in plasma volume by as much as 25%. He (138) further noted that the decrements in PV were accompanied by significantly reduced endurance capacities and maximal blood lactate while cardiac output may be maintained by increased heart rate. Costill and Fink (36) compared the effects of thermal- and exercise-induced hypohydration, and reported that at 4% body weight loss PV was reduced by 16-18% subsequent to either form of dehydration. In our own experiments induction of hypohydration (3-7% body weight) is ordinarily followed by a period (12-16 h) of inactivity before experimentation during which fluid is redistributed to the plasma, yet PV's consistently decrease by 5-15% (139-141).

When we dehydrated men by 5% bw, we observed significant elevations in PRA and PA in blood samples taken prior to exercise; subsequent exercise in the heat reduced PV even further and PRA and PA were enhanced during hypohydration trials while the increases in PRA were modulated by acclimation (64) (Fig. 4). In a more recent study (65) in which we reported the effects of 3,5, and 7% hypohydration on circulating hormone responses, we observed that increasing the intensity of hypohydration from 3% to 5% was usually accompanied by an increased elevation in PRA and PA; however, between 5 and 7% hypohydration, where no further decrements in PV were observed (141), we likewise noted no additional increments in either PRA or PA (Fig. 5). Consistent with these results Von Ameln et al. (165) demonstrated that, following thermal dehydration, PVP increased from 2.1 to 8.1 pg/ml, but this elevation was attenuated to 4.7 pg/ml by head-out water immersion. They attributed this attenuation of PVP response to "central hypervolemia" effected by water immersion.

When the same group (101) examined the effects of hypohydration induced by prolonged exercise (27-32 km) on PRA, they reported that four-fold increases in PRA levels 50-60 min after the completion of exercise were reduced to nearly control levels about 1 h later and following food and fluid consumption. In a separate experiment (16) subjects were exercised in the heat (34°C, 85 W, 4 h) with no fluid replacement, with water replacement, and with NaCl-sucrose consumption (Fig. 6). During the exercise interval levels of PA, PRA, and PVP were progressively increased during the "no fluid" trial; however, with either water or NaCl-sucrose replacement, increments in PRA and PVP were abolished. While water consumption attenuated the increment in PA elicited by no fluid replacement, NaCl-sucrose blunted this response even further, and after 4 h of exercise in the heat, levels were not different from controls.
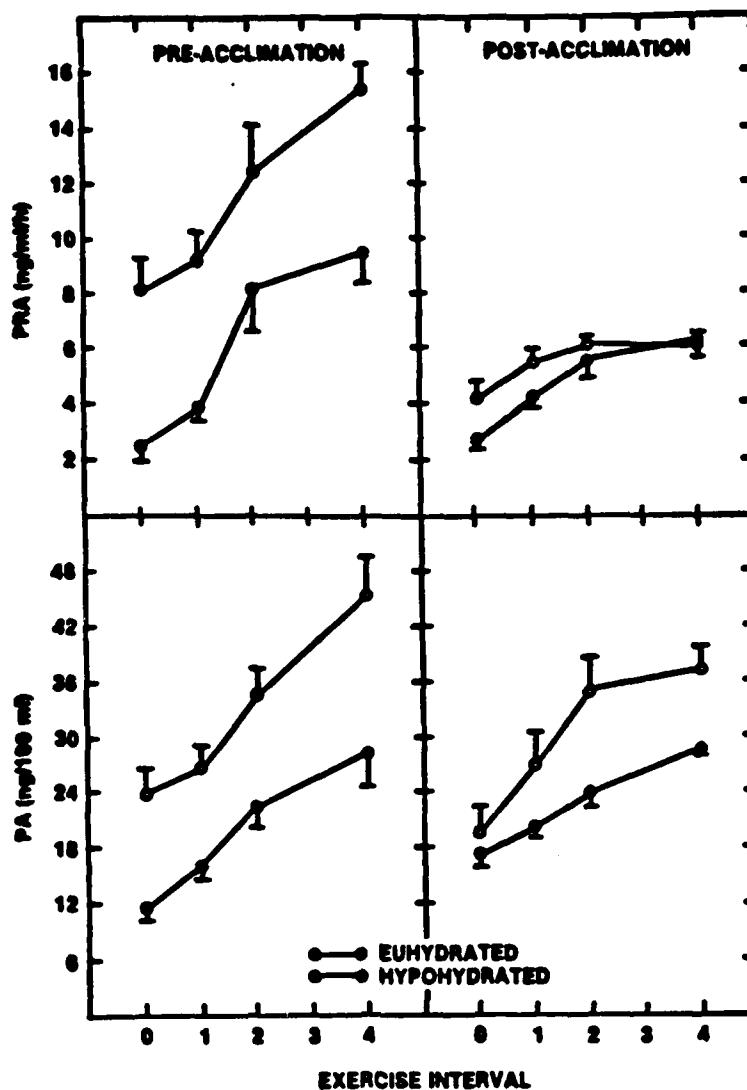
Fig. 4 Effects of hypohydration (5% bw) and acclimation on plasma renin activity and aldosterone levels during exercise (four 25 min/10 min work/rest cycles, level treadmill, 1.34 m/sec) in the heat (49°C, 20% rh). Mean levels (± SEM) are depicted for 8 young adult male and 8 young adult female test subjects. Blood was taken prior to and approximately 15-20 min into the 1st, 2d, and 4th exercise intervals. The increased plasma volume of acclimation may help to maintain renal blood flow and repress the PRA response. The effects of hypohydration are most notable pre-acclimation (time 0). (Redrawn from: Francesconi et al., J. Appl. Physiol., 55, 1790, 1983 with permission of the publisher)

Thus, the evidence seems to favor strongly a rapid and marked elevation in circulating levels of fluid- and electrolyte regulatory hormones when heat/exercise exposure induces progressive dehydration or a targeted level of hypohydration precedes the heat/exercise exposure. These responses may be related to increased osmolality, decreased plasma volume, or perhaps decreased blood pressure. While the responses of PVP and PRA have been consistent and unidirectional, there is some evidence that the control of aldosterone secretion may be superseded by the increasing Na^+ concentration which also may accompany hypohydration. In fact, reports on the influence of electrolyte supplements during heat exposure and exercise in the heat, the effects of prior high or low dietary consumption of electrolytes, and the experimental depletion of electrolytes provide a framework to investigate further the mechanisms and control of hormonal responses to heat/exercise stress.
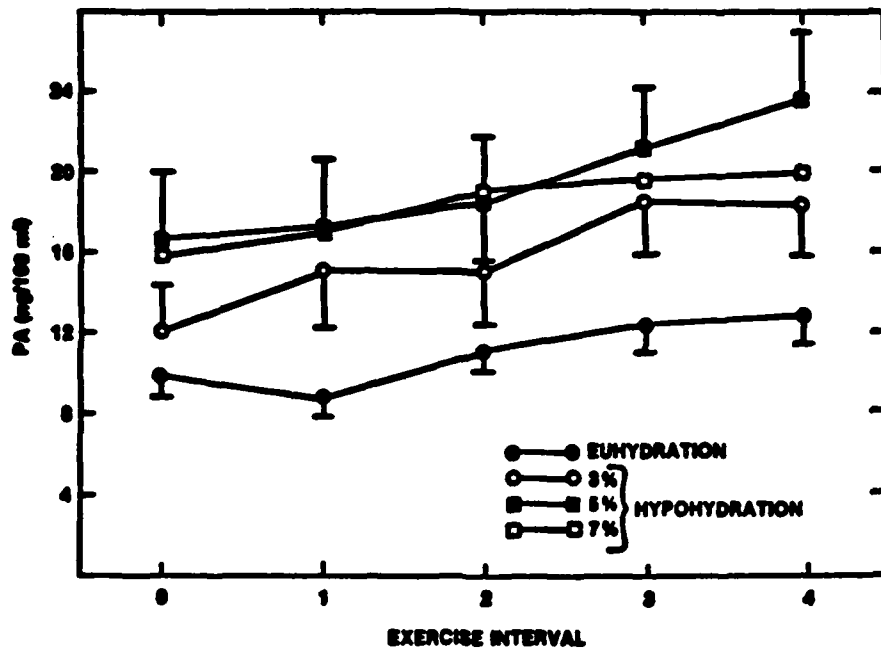
Fig. 5 Effects of graded hypohydration levels (0-7%) on PA during exercise in the heat in 6 highly acclimated young adult male test subjects. Mean values ± SEM are depicted. Hypohydration to the appropriate level was accomplished by fluid denial during exercise in the heat during the afternoon prior to the morning of the test. The exercise intervals correspond to 4 consecutive exercise periods (25 min, level treadmill, 1.34 m/sec, 49°C) interspersed by 10 min rest periods. Time 0 blood samples were taken after sujects remained upright in an antechamber (20°C) for at least 20 min. Five and 7% hypohydration elicit similar elevations in resting PA levels; it is noteworthy that PV were reduced to similar degrees by 5 and 7% hypohydration indicating the role of volume receptors in controlling the endocrine responses. (Redrawn from: Francesconi et al., J. Appl. Physiol. 59, 1855, 1985 with permission of the publisher)

ELECTROLYTES

In his review on fluid and electrolyte balance during exercise in hot environments, Halim (80) concluded that 3 g of salt should ordinarily be ingested for each liter of sweat loss. If the exercise intensity and the ambient temperature are sufficiently high, then it is not unusual that 6-12 L of sweat/day may be secreted requiring, accordingly, 300-600 mEq NaCl for replacement. However, in their more recent review, Epstein and Sohar (46) surveyed a variety of reports in the literature which suggested that well-acclimated individuals can tolerate work in the heat with NaCl intakes even less than 100 mEq/day. Of course, several early reports had confirmed that the acquisition of acclimation is accompanied by a striking ability to secrete increased quantities of a more dilute sweat (39,130,131,162). The requirement for salt replacement during repeated or prolonged exercise in hot environments is dependent upon the individual's acclimation status as well as the normal amount of dietary salt to which the test subject or population may be accustomed.
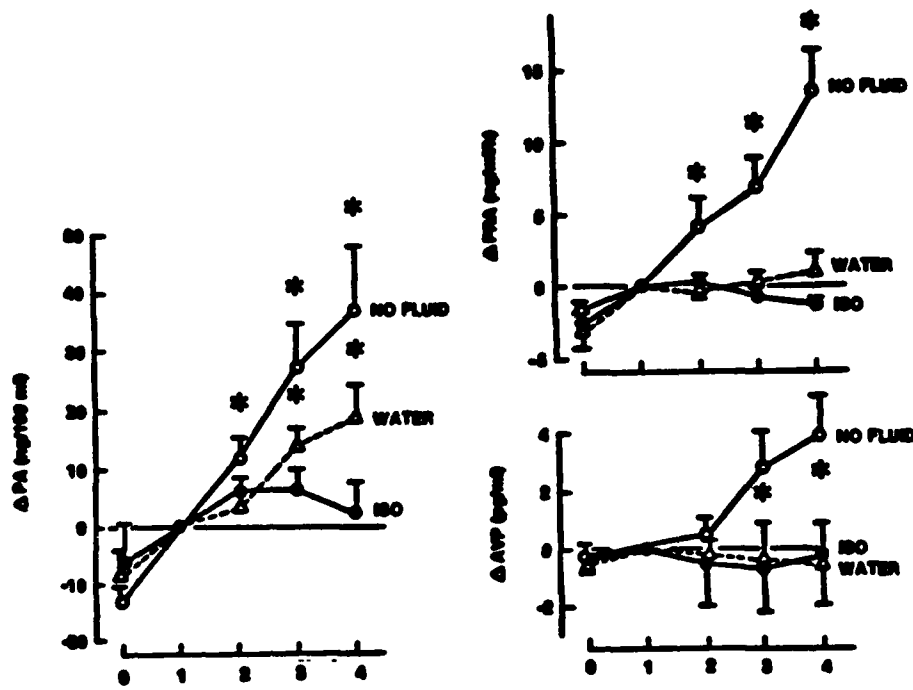
Fig. 6 Effects of intermittent exercise in the heat (34°C) on plasma renin activity, aldosterone, and vasopressin. Exercise consisted of four 25 min/5 min work/rest cycles followed by four 20 min/10 min work/rest cycles at approximately 85W. Three separate trials were evaluated: NO FLUID, exercise was accomplished with no fluid intake during the trial; WATER, 80% of the weight loss was replaced with spring water; ISO, 80% of the weight loss was replaced with an isotonic solution of sodium, chloride, potassium, and sucrose. Mean values (+SEM) are depicted for a group of 5 unacclimated young men. Asterisks indicate significant difference from the 1 h value (p<0.05). (Redrawn from: Brandenberger et al., Eur. J. Appl. Physiol., 55, 123, 1986 with permission of the publisher)

When Smiles and Robinson (157) examined the effects of recurrent exercise in the heat (100 min, 1.58 m/s, 45°C) on young adult males in negative salt balance induced by dietary manipulation, they reported 3-6 fold increments in 24 h output of urinary tetrahydroaldosterone; this was accompanied by apparent 10-20-fold reductions in urinary Na^+. When the same experiments were repeated with the subjects in positive salt balance, both urinary tetrahydroaldosterone and Na^+ were at control levels suggesting that the magnitude of the sodium conservation response during acclimation is dependent upon exogenous Na^+ availability. Shortly thereafter, Bailey and co-workers (10) placed 12 young adults males on high (>300 mEq/d), normal (182 mEq), and low (12 mEq) Na^+ diets during 3 consecutive weeks. Following the normal and low dietary intervals, heat stress was induced by sedentary exposure to 46-51°C ambient conditions and PA and PRA were assessed before and after the heat stress. The low-Na^+ diet elicited significant increments in both PA and PRA in the pre-heat samples, and the heat exposure increased the levels of both hormones following either dietary regimen though the elevations were more notable during the low salt trial. Interestingly, they noted that packed cell volumes were unaffected by thermal stres, and concluded that high thermal stress, per se, is a potent stimulator of renin and aldosterone secretion. In fact, these workers suggested that the level of heat stress elicited by sedentary exposure to approximately 50°C might be utilized as a marker of the normal response of these endocrine systems (10).

Recent results have generally indicated that exogenous salt loading does not markedly enhance the acquisition of acclimation or reduce the physiological strain of work in the heat. Although Armstrong et al. (6) observed several sporadic advantages to consuming a high sodium diet during acclimation to heat, Konikoff et al. (107) reported that salt supplementation in fully acclimated subjects provided no benefits during work in the heat

while PA levels were repressed and PRA was unaffected. Earlier, Follenius et al. (54) had demonstrated that consumption of a low salt diet induced significant increments in basal levels of both PA and PRA; when these subjects were acutely exposed (90 min) to sedentary heat stress, the percent increases in PA and PRA were similar in subjects consuming either a high salt (200 mEqNa/d) or low salt (20-30 mEqNa/day) diet. The same group (17) used propranolol to illustrate a dissociation between the responses of PA and PRA to heat stress in that propranolol reduced the PRA response to heat exposure while PA was actually increased during the propranolol trial. We have observed dissociations in PRA and PA responses previously in both rats (57) and, under certain conditions, humans (65).

Likewise, when studying rehydration with a glucose-electrolyte solution or water, Costill et al. (35) concluded that, following dehydration to 3% body weight on 5 successive days, the glucose-electrolyte drink did not provide any physiological benefits to the test subjects. The authors cautioned, however, that electrolyte balance was being maintained by adequate dietary consumption. In fact, they hypothesized (35) that the supplementary Na^+ intake may have repressed PA secretion causing more Na^+ and fluid loss during the glucose-electrolyte trial. In addition, Nielsen et al. (119) recently reported no significant effects of rehydration with a control, high potassium, high sodium, or high sugar beverage; interestingly, they concluded that the high Na^+ drink specifically benefitted the extracellular fluid compartment while the high K^+ and high sugar drinks augmented intracellular fluid volume. Ikawa et al. (96) had previously exposed volunteers to a sauna (65-70°C) for 30 min and during a recovery period provided either no fluids, 500 ml of an isotonic sports supplement containing Na^+, or 500 ml of water. Circulating A levels were significantly increased by the heat exposure, and were maintained at higher concentrations during the water or no-fluid experiment. The various drinks did not provide any advantages in reducing physiological strain with respect to either rectal temperature or heart rate.

The evidence is weighted heavily toward the conclusion that in the presence of consistent dietary intake of salt, excessive salt consumption or the ingestion of salt-containing beverages does not have ergogenic effects during exercise in the heat or reduce the physiological cost of heat exposure. Harrison et al. (84) noted that when 1% NaCl was ingested to prevent dehydration, core temperatures were higher than when water was consumed. In fact, there is increasing evidence to suggest that consumption of modest or even low levels of salt (<100 mEq/d) is adequate to maintain electrolyte balance when the reduced dietary intake is followed by acute or prolonged heat exposure or exercise in the heat. Further, the maintenance of electrolyte balance under these conditions is dependent upon the effects of increased endocrinological activity as manifested in consistently elevated PA concentrations when dietary NaCl intake is low, and reduced circulating PA when NaCl intake is above normal.

Several of the world's population groups are able to withstand chronic heat exposure on low salt diets (20-50 mEq/day) without apparent increased rates of heat injury; this is probably testimony to the exquisite mechanisms by which humans adapt endocrinologically for extremely efficient salt conservation. From this it may be reasonable to conclude that individuals who are at most serious risk of developing salt depletion-induced heat cramps (14) are those who abruptly change from a high or average salt intake to a low consumption concomitant with sustained heat exposure and profuse sweating. In our experience we have observed this situation when large numbers of garrison quartered troops are abruptly transferred to desert environments in the southwestern US for field training exercises.

The effects of potassium (K) deficiency upon the ability to work in the heat and the endocrinological responses to acute or chronic heat exposure have been less extensively studied. Malhotra et al. (113) reported that K

deficiency can accrue even in well-acclimated individuals when exposed sedentarily to 40°C for 4 consecutive days, and, further, that this K deficiency may contribute significantly to the development of heat injury. In fact, during acclimation to heat it is conceivable that the loss of K in sweat as well as the conservation of sodium by the kidney with attendant K loss could contribute to cellular potassium depletion and the development of heat illness (142).

Knochel and his co-workers (103,104) have demonstrated that recurrent work in hot environments may result in substantial negative K balance despite mean daily intakes of approximately 100 mEq; these investigators also observed that both PA and PRA, as well as urinary aldosterone were "unsuppressed" for the quantities of Na that their test subjects were ingesting although no explanation for the lack of suppression was offered. Alternatively, when Coburn et al. (21) acutely exposed unacclimated, K-deficient test subjects to heat stress, they observed repressed urinary aldosterone excretion which they attributed to a failure of plasma K to increase during heat exposure as a result of the K depletion. Interestingly, their regimen of K depletion (producing deficits of 230-465 mEq K) failed to induce consistent thermoregulatory decrements in their test subjects. When Francis and co-workers (66,67) used either water or a potassium supplemented electrolyte solution for rehydration during exercise (50% VO_2 max, 120 min, 32°C), they observed no marked beneficial effects of the electrolyte solution; PRA and PA were significantly reduced during the electrolyte supplement experiment. Generally, the consensus has been that carbohydrate/electrolyte supplements are unnecessary if balanced food consumption has not been curtailed (110).

When Hubbard et al. (95) used their rat model of human heatstroke (93,94) to investigate the effects of prolonged feeding (32 d) of a K deficient diet, they reported that exercise endurance was reduced by 37%, work done was decremented by 49%, and muscle K was depleted by 26%. At approximately the same time Haldy and Muller (79) had reported, also in rats, that consumption of a K-deficient diet (2 weeks, 0.8 mmole K/kg) markedly reduced the ability of excised and incubated adrenal glands to convert tritiated corticosterone to aldosterone. Since the biosynthesis of aldosterone in K-deficient rat adrenal glands could be stimulated by sodium deficiency, water deprivation, and furosemide treatment, these authors concluded (79) that enzymes regulating the final steps in the biosynthesis of aldosterone may be instrumental in the control of PA levels and inhibited by potassium deficiency.

In his review Knochel (102) concluded that K deficits may be associated with postural hypotension and syncope, reduced muscle membrane integrity, and the development of rhabdomyolysis. While frank intracellular hypokalemia will clearly predispose an individual to increased risk of heat injury, other potential sequelae of acute or chronic negative K balance have not been assessed in carefully controlled human studies. Interestingly, in our studies on heatstroke in rats we have demonstrated that the intensity of circulatory hyperkalemia subsequent to exercise in the heat was inversely correlated to survival time (60); conversely, rats which were able to run for prolonged periods with the achievement of a steady-state, moderate Tre (<40°C) manifested reduced circulating K levels (59). Clearly, the relationships between K deficits and surfeits and plasma volume, sweat secretion, hormonal responses, Na balance, and the acquisition of acclimation in humans should be more extensively described (7).

HEAT ACCLIMATION

The effects of heat acclimation on the endocrinological responses to heat exposure/exercise in the heat are somewhat inconsistent and variable. When considering the number and range of experimental variables which could conceivably affect such responses, this is perhaps not surprising. For

example, hydration level, acclimation status, aerobic fitness level, exercise intensity and type, ambient conditions, electrolyte balance, sampling time, subject posture and a variety of other experimental variables (19) could, singly or in combination, affect test results and conclusions. Of these, the acclimation state may especially provide significant variability due to the range of increments in resting plasma volume which has been consistently reported subsequent to heat acclimation.

When Greenleaf et al. (73, 75) exercised (45% VO_2 max) 2 groups of men at 23.8°C and 39.8°C for 8 d, they reported that during exercise in the heat, PRA and PVP levels were increased without effects of acclimation on the resting levels prior to or increments during exercise in the heat (Fig. 7). Likewise, when Finberg et al. (51) measured PRA and PA following exercise (ergometer, 40-50% VO_2 max, 30 min) in the heat (50°C) for 7 consecutive days, they reported similar PRA (9.5 $\pm$ 4.4 and 8.0 $\pm$ 4.7, day 1 vs. day 7) and PA (22.6 $\pm$ 8.5 and 25.5 $\pm$ 8, day 1 vs. day 7) levels prior and subsequent to
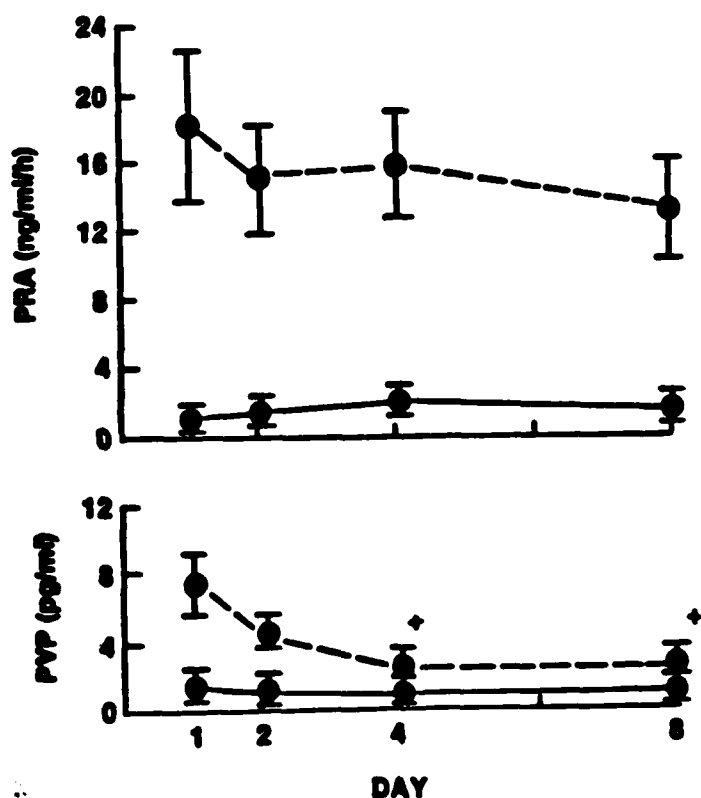

DAY

Fig. 7 Effects of heat acclimation (39.8°C, 2 h/d, 8 d, ergometer, 75W) on plasma renin activity and vasopressin levels immediately prior (solid line) and subsequent (dashed line) to exercise in the heat. Venous blood samples were taken on d 1,2,4, and 8 and the data represent mean levels for 5 healthy young males. The trend toward reduced PRA over the 8 d acclimation regimen did not achieve statistical significance although the data for PVP can be interpreted in terms of an increase in PV developing during acclimation. The continuous 2h exercise period may have contributed to the persistently high PRA even after acclimation. (+ = significantly different from day 1) (Redrawn from: Greenleaf et al., J. Appl. Physiol., 54, 414, 1983 with permission of the publisher)

the acclimation regimen. Bonner et al. (15) had earlier reported similar elevations in PA prior and subsequent to acclimation and following both a sedentary and exercise interval in the heat. Davies and co-workers (22,37) concluded that an 11 day acclimation program did not attenuate the increments in PRA and PA observed during exercise in the heat, but prior saline ingestion did modulate these increments suggesting the more subtle effects of acclimation vs saline consumption on endocrinological responses. When Convertino and Kirby (32) observed a significant reduction in 24 h Na clearance following acclimation with no changes in resting PA levels, they concluded that renal sensitivity to PA may be increased during acclimation.

A short time later the same workers (100) demonstrated reductions in sweat sodium loss concomitant with reductions in PA during exercise in the heat (45% VO_2 max, 40°C) on day 10 vs day 1 of heat acclimation. Again, they hypothesized that the reduced PA is compensated by an elevated eccrine sweat gland responsivity to PA after acclimation. The pioneering work in this area had been accomplished much earlier by Conn who reported that acclimation can reduce the salt lost in sweat by as much as 95% (27). Finberg and Berlyne (50) had used both natural acclimatization (exposure to summer heat) and experimentally induced heat acclimation (90 min exercise, 50°C, 7 d) to demonstrate generally attenuated responses of PRA and PA to light exercise in the heat when compared to the responses elicited in winter or without acclimation. They (52) had earlier reported that not only natural acclimatization to summer heat, but also prehydration with a NaCl solution suppressed exercise/heat-induced increments in PRA.

Some of our own work (64,65) had also demonstrated attenuated responses of the fluid/electrolyte-regulatory hormones to exercise in the heat following heat acclimation. When Shvartz et al. (156) administered tilt-table tests prior and subsequent to heat acclimation and observed 50 and 75% decrements, respectively, in responses of PRA and PVP following acclimation, they ascribed these decreases to the increased resting plasma volume. Alternatively, when we acutely expanded PV by intravenous administration of hyperoncotic albumin, we reported that during exercise (63) or at rest (58) in the heat, PA levels were suppressed. Thus, the augmented cardiovascular stability (137, 167) and plasma volume (151, 166) of heat acclimation may combine to lessen the requirement and stimulus for the usual elevations in levels of fluid and electrolyte regulatory hormones during heat exposure or work in the heat.

Knochel and Vertel (105) hypothesized that the development of the acclimated condition is characterized by increased aldosterone secretion and excretion. Earlier, Fletcher et al. (53) had compared aldosterone excretion rates in residents of the United Kingdom (15-17°C) and British expatriates living in Kuwait (38°C) and reported mean 24 h urinary aldosterone levels of 8.7 and 13.4 ug, respectively. Knochel and Vertel (105) reasoned that the increased aldosterone secretion and excretion of heat acclimation could effect excessive urinary K loss and suggested that less vigorous NaCl replacement (especially in the form of salt tablets) may be appropriate in reducing electrolyte disorders, polyuria, and circulatory hypokalemia.

However, since these earlier suggestions that increased aldosterone secretion and activity may be necessary for the acquisition of heat acclimation (53,68,105), more recent evidence indicates that acclimation and aerobic exercise training (117) are accompanied by an increased sensitivity and responsivity to endocrinological effects at the sites of both central control (118,129) and peripheral effectors (32,100,151). The majority of the data suggests that subjects who are fully heat acclimated respond to heat exposure/exercise in the heat with similar or attenuated levels of PVP, PA, and PRA when compared to their non-acclimated counterparts. Such responses appear to be consistent with the increased plasma volume, decreased physiological strain, increased perfusion of viscera, and electrolyte balance associated with heat acclimation.

The thermoregulatory and cardiovascular advantages of increased plasma volume and total body water in reducing the physiological strain of exercise in the heat have been investigated and recognized for decades. Moroff and Bass (115) used the same test subjects in two experimental trials (90 min treadmill exercise, 49°C, 1.58 m/s), once after preingestion of 2000 ml water and once without preingestion. They reported (115) that hyperhydration resulted in lower Tre, heart rate, and increased sweat secretion during exercise in the heat. Much later, Fortney et al. (56) studied the effects of hypovolemia (diuretic-induced) and hypervolemia (albumin infusion) on blood volume and sweating responses. They observed that hypovolemia (8.7% decrease in blood volume) and hypervolemia (7.9% elevation in blood volume) effected 20 min sweat rates of 270 ml and 541 ml, respectively (56), and hypothesized that the exacerbated release of PVP during hypovolemia may have contributed to this decrement. Earlier, Horstman and Horvath (91) reported that whole body, thigh, and abdomen sweat rates were significantly greater during euhydration than 3.6% hypohydration. Alternatively, Myhre and Robinson (116) used passive heat exposure (50°C) to reduce PV by 7.8% (no fluid replacement) or 2.9% (NaCl replacement) and demonstrated no effects on sweat rates; in these experiments the PV differential may have been insufficient to elicit observable differences (87). In a related experiment Shannon et al. (154) exercised men in a cool (15°C) environment, once after a subcutaneous administration of VP and a second time when subjects ingested 2% of their body weight in water after VP injection. Thirty through 60 min after the initiation of exercise, PV loss was reduced in the hyperhydrated group indicating the important effects of hydration level even when exogenous VP is administered (154).

The mechanisms responsible for the increased PV of heat acclimation have been extensively investigated in recent years. Thus, Senay and co-investigators (152) exercised trained individuals (4 h/d, 40-50% VO_2 max, 3 d at 25°C and 10d at 45°C), and reported that between d 1 and d 2 of exercise in the heat total plasma protein was elevated by 11.6% and PV increased by 9%. Further, during acclimation more protein and fluid were retained within the circulatory system (152). Earlier, Senay (145) had compared the effects of stair-stepping in a cool environment (20°C) with the same exercise in the heat (40°C), and reported that following acclimation, exercise in the heat was accompanied by hemodilution and maintenance or increments in plasma protein. Conversely, before heat acclimation exercise at 40°C was characterized by hemoconcentration and net protein losses (145) while exercise in the cool environment even before heat acclimation was accompanied by hemodilution. These results led Senay (145, 147) to hypothesize that permeability changes in cutaneous capillaries and availability of translocatable protein were partially responsibile for the increased PV of heat acclimation.

When Senay later (146) compared the effects of heat acclimation on block-stepping at 34°C, he again found that with acclimation, hemodilution was maintained for 2 h. He further hypothesized (146) that hemodilution or hemoconcentration responses may be dependent upon initial plasma osmolalities and circulating VP levels with the degree of hemodilution inversely correlated with initial osmolality and VP concentration. Interestingly, the same group (150) asserted that heat intolerant individuals (n=15) failed to hemodilute to the same extent as a group of heat tolerant men (n=19) when matched for VO_2 max, under similar ambient and work conditions. Moreover, reduced aerobic exercise in the heat or deacclimation was accompanied by a decrement in plasma volume mostly accounted for by significant reductions in total plasma protein (126). Even in rats, Horowitz and Samueloff (90) reported that dehydration following acclimation to 34°C ambient was characterized by decreased efflux of albumin while colloid osmotic pressure and total protein mass increased. Studying desert-originated mice, they demonstrated (89) that dehydration elicited no changes in plasma volume and a significant decrement in efflux of albumin from the circulatory system.

Table 1. Compendium of data from representative reports. Note that in some cases quantitative estimates were from interpretations of figure-depicted data.

LIST OF ABBREVIATIONS

PRA = PLASMA RENIN ACTIVITY; VO_2 MAX = MAXIMAL OXYGEN CONSUMPTION; PADH = PLASMA ANTIDIURETIC HORMONE; NA = SODIUM; μU = MICROUNITS; PRE-EX = PRE-EXERCISE; POST-EX = POST-EXERCISE; H = HOURS; D = DAYS; M = METERS; ACCL = ACCLIMATIZED; PA = PLASMA ALDOSTERONE; HYPO = HYPOHYDRATED; ng = NANOGRAM; pg = PICOGRAM; PVP = PLASMA VASOPRESSIN; DEP = DEPRIVED; REHY = REHYDRATION; TRE = RECTAL TEMPERATURE; RT = (NORMAL) ROOM TEMPERATURE; W = WATTS; RH = RELATIVE HUMIDITY, KPM = KILOPOND METERS

CITATION	SPECIES	ACTIVITY	CONDITIONS AND RESULTS
GROZA ET AL., PHYSIOLOGIE 14, 71, 1977	RAT	SEDENTARY	22°C, PADH = 3 μU/ml; 2H, 40°C, PADH = 6.75 μU/ml; 2H, 40°C, 5MG/KG PROPRANOLOL PADH = 10.5 μU/ml
FRANCESCONI ET AL., J. APPL. PHYSIOL. 58, 152, 1985	RAT	SEDENTARY	21°C, PA = 0.5 ng/ml; 35°C, PA = 0.5 - 1.1 ng/ml; 71D, LOW NA DIET, 21°C PA = 3.9 ng/ml; 71D, LOW NA DIET, 8-10H 35°C, PA = 6 - 17 ng/ml
FRANCESCONI ET AL., J.APPL.PHYSIOL. 55, 876, 1983	RAT	EXERCISE, TREADMILL 9.14/MIN	22°C PRE-EX, PA = 0.4 ng/ml; POST-EX, 35°C, PA = 0.9 ng/ml; 22°C, PRE-EX, LOW NA DIET 57D, PA = 1.87 ng/ml; POST-EX, 35°C, 57D LOW NA DIET, PA = 1.69 ng/ml
ARAD ET AL.; J.COMP.PHYSIOL 155, 227, 1985	FOWL	SEDENTARY	26°C, PADH = 9.5 pg/ml, PA = 6.9 pg/ml; 42°C, ACCL, PADH = 19 pg/ml, PA = 11 pg/ml; 26°C, 13.4% K+PO, PADH = 17 pg/ml, PA = 11 pg/ml; 42°C ACCL, 13.4% HYPO, PADH = 21 pg/ml, PA = 24 pg/ml
THRASHER ET AL., AM.J.PHYSIOL. 247, R76, 1984	DOG	SEDENTARY	22°C, PVP = 2.6 pg/ml, PA = 5.0 ng/100ml; 22°C, 24H WATER DEP, PVP = 8 pg/ml; 22°C, 24H WATER DEP, 24H REHY, PA = 8ng/100ml
SZCZEPANSKA-SADOWSKA ET AL., AM.J.PHYSIOL. 226, 155, 1974	DOG	SEDENTARY	20-25°C, PADH = 2.4 μU/ml; HEAT BRAIN 1.5°C, PADH = 24 μU/ml; HEAT HYPOTHALAMUS 2°C, PADH = 200 μU/ml
EL-NOUTY ET AL., J.APPL.PHYSIOL. 48, 249, 1980	CATTLE	SEDENTARY	20°C, PADH = 1.0-1.5 pg/ml; 35°C, PADH = 1.7-2.8 pg/ml; 35°C, WATER DEP 30H, PADH = 2.5-9.0 pg/ml

Reference	Species	Condition	Results
EISMAN ET AL., J.APPL.PHYSIOL. 43, 739, 1977	BABOONS	SEDENTARY (RESTRAINT)	25°C-38°C, NO CHANGE PRA; 42.5°C-49°C, PRA UP 97.5%/°C INCREASE Tre; PROPRANOLOL PREVENTED INCREASED PRA
GIBINSKI ET AL., EUR.J.APPL.PHYSIOL 42, 1, 1979	HUMAN	SEDENTARY	RT, PADH = 3.9 µU/ml; 1H, 43°C PADH = 17.7 µU/ml; 2H, 43°C PADH = 16.2 µU/ml
BAILEY ET AL., EXPERIENTIA 28, 159, 1972	HUMAN	SEDENTARY	RT, NORMAL NA INTAKE PA = 13 ng/100ml, PRA = 2.8 ng/ml/h; RT, LOW NA INTAKE PA = 24.3 ng/100ml, PRA = 16.5 ng/ml/h; 46-51°C, NORMAL NA INTAKE PA = 22.8 ng/100ml, PRA = 6.3 ng/ml/h; 46-51°C, LOW NA INTAKE PA = 44.8 ng/100ml, PRA = 18.5 ng/ml/h
FOLLENIUS ET AL., EUR.J.APPL.PHYSIOL. 41, 1979	HUMAN	SEDENTARY	28°C, LOW NA INTAKE PA = 11-29 ng/100ml, PRA = 3-3.5 ng/ml/h; 28°C, HIGH NA INTAKE PA = 5-7 ng/100ml, PRA = 2-2.5 ng/ml/h; 46°C, LOW NA INTAKE PA = 18-35 ng/100 ml, PRA = 3.5-6.5 ng/ml/h; 46°C, HIGH NA INTAKE PA = 5-12 ng/100 ml, PRA = 2.5-3.5 ng/ml/h
FOLLENIUS ET AL., HORM.METAB.RES. 11, 180, 1979	HUMAN	SEDENTARY	28°C, PA = 10-19 ng/100 ml; 46°C, PA = 18-38 ng/100 ml
ESCOURROU ET AL., J.APPL.PHYSIOL 52, 1438, 1982	HUMAN	SEDENTARY	RT, PRA = 182 ng/100ml/3h; TRE INCREASE 1°C WATER-PERFUSED SUIT, PRA = 289 ng/100 ml/3h; PROPRANOLOL = INCONSISTENT EFFECTS ON PRA
BRANDENBERGER ET AL., J.ENDOCRINOL.INVEST. 4, 395, 1980	HUMAN	SEDENTARY	28°C, LOW NA INTAKE, PA = 15-22 ng/100 ml, PRA = 4-5 ng/ml/h; 46°C, LOW NA INTAKE, PA = 15-37 ng/100 ml, PRA = 4.5-15 ng/ml/h; 28°C, PROPRANOLOL, LOW NA INTAKE, PA = 15-35 ng/100ml, PRA = 2-3 ng/ml/h; 46°C, PROPRANOLOL, LOW NA INTAKE, PA = 15-63 ng/100 ml, PRA = 2-7 ng/ml/h
ROCKER ET AL., EUR.J.APPL.PHYSIOL 49, 69, 1982	HUMAN	SEDENTARY	19-21°C, PADH = 3.5 pg/ml; 70-76°C, PADH = 9.8 pg/ml; 90 MIN AFTER 70-76°C, PADH = 6.5 pg/ml
DUMOULIN ET AL., COMPTES RENDUS 174, 146, 1980	HUMAN	SEDENTARY	RT, PA = 143 pg/ml, PRA = 2.1 ng/ml/h; 80°C, PA = 207 pg/ml, PRA = 6.8 ng/ml/h

Reference	Species	Condition	Data
KOSUNEN ET AL., J.APPL.PHYSIOL. 41, 323, 1976	HUMAN	SEDENTARY	RT, PA = 149 pmole/l, PRA = 1.9 ug/l/h; 86°C-90°C PA = 769 pmole/l, PRA = 3.85 ug/l/h
BONNER ET AL., J.APPL.PHYSIOL. 41, 708, 1976	HUMAN	SEDENTARY, 155 MIN, EXERCISE, 50W, 30MIN ERGOMETER	28°C, PRE-ACCL, PA = 10.6ng/100ml, POST-ACCL, PA = 8.9 ng/100ml; 48°C, PRE-ACCL, AFTER 155 MIN HEAT PA = 12.8 ng/100 ml, POST-ACCL, PA = 11.1 ng/100ml; 48°C, PRE-ACCL, 30 MIN POST-EX, PA = 26.7 ng/100 ml, POST-ACCL PA = 24.8 ng/100 ml
KIRBY ET AL., J.APPL.PHYSIOL. 61, 967, 1986	HUMAN	EXERCISE, 46% VO_2 MAX, ERGOMETER	49°C, DAY 1, 1H EX, PA = 75 ng/100ml, 2H EX, PA = 135 ng/100 ml; 49°C, D10, 1H EX, PA = 50ng/100ml, 2H EX, PA = 90ng/100ml
GREENLEAF ET AL., J.APPL.PHYSIOL. 54, 414, 1983	HUMAN	EXERCISE 75W ERGOMETER	24°C, DAY 1, POST-EX PRA = 6ng/ml/h, PVP = 2 pg/ml; 49°C, DAY 1, POST-EX, PRA = 18 ng/ml/h, PVP = 8 pg/ml; 24°C, DAY 8, POST-EX, PRA = 2.5 ng/ml/h, PVP = 2 pg/ml; 49°C, DAY 8, POST-EX, PRA = 14 ng/ml/h, PVP = 8 pg/ml
FINBERG ET AL., ISRAEL J.MED.SCI. 12, 844, 1976	HUMAN	EXERCISE ERGOMETER, 450kpm/min	PRE/EX PRA = 1.6 ng/ml/h, PA = 5.4 ng/100ml; 26°C POST-EX, PRA = 1.2 ng/ml/h, PA = 10.2 ng/100ml; 58°C, POST-EX, PRA = 5.7 ng/ml/h, PA = 22.6 ng/100ml
FINBERG ET AL., ISRAEL J.MED.SCI. 12, 1638, 1986	HUMAN	EXERCISE, ERGOMETER 46% VO_2MAX	POST-EX, D1, PRA = 9.5 ng/ml/h, PA = 22.6 ng/100ml; POST-EX, D7, PRA = 8.0 ng/ml/h, PA = 25.6 ng/100ml
PAOLONE ET AL., MED.SCI.SPORTS.EXER. 15, 97, 1983	HUMAN	EXERCISE, ERGOMETER, 54% VO_2MAX	25°C, YOUNG MEN PRE-EX, PRA = 1.48 pmole/ml/h, POST-EX, PRA = 3.05 pmole/ml/h; 35°C OLDER MEN, PRE-EX, PRA = 1.36 pmole/ml/h, POST-EX, PRA = 2.59 pmole/ml/h; 35°C, YOUNG MEN, PRA = 3.93 pmole/ml/h, 60 MIN EX, PRA = 5.91 pmole/ml/h 20 MIN EX
CONVERTINO ET AL., J.APPL.PHYSIOL 50, 123, 1981	HUMAN	EXERCISE, ERGOMETER, 100,175, AND 225W	RT, PRE-EX PRA = 2 ng/ml/h, PVP = 1 pg/ml; RT, POST-EX, 100W PRA = 3.5 ng/ml/h, PVP = 2 pg/ml; RT, POST-EX, 175W PRA = 5.5 ng/ml/h, PVP = 7 pg/ml; RT, POST-EX, 225W PRA = 7.5 ng/ml/h, PVP = 21 pg/ml

Reference	Species	Exercise	Results
CONVERTINO ET AL., J.APPL.PHYSIOL. 54, 508, 1983	HUMAN	EXERCISE, ERGOMETER, 100, 175, AND 225W	PRE-TRAINING, POST-EX, 175 W, PRA = 6.8 ng/ml/h, PVP = 3.7 pg/ml; POST-TRAINING, POST-EX, 175W, PRA = 4.4 ng/ml/h, PVP = 1.5 pg/ml; PRE-TRAINING, POST-EX, 225W, PRA = 15.3 ng/ml/h, PVP = 8.3 pg/ml; POST-TRAINING, POST-EX, 225W, PRA = 0 ng/ml/h, PVP = 0 pg/ml
BRANDENBERGER ET AL. EUR.J.APPL.PHYSIOL. 55, 123, 1986	HUMAN	EXERCISE ERGOMETER 85W	4H WORK, NO FLUID PRA UP 14 ng/ml/h, PA UP 37 ng/100ml, PVP UP 4 pg/ml; 4H WORK REPLACE FLUID LOSS WITH WATER, PRA UNAFFECTED, PA UP 19 ng/100ml, PVP UNAFFECTED; 4H WORK, REPLACE FLUID LOSS WITH NUTRIENT SOLN, PRA UNAFFECTED, PA UNAFFECTED, PVP UNAFFECTED
GREENLEAF ET AL., J.APPL.PHYSIOL. 51, 298, 1981	HUMAN	EXERCISE ERGOMETER 44% VO2max	24°C, D1; PVP = 2.0 pg/ml; PRA = 1.0 ng/ml/h; 24°C, D9; PVP = 2.0 pg/ml; PRA = 1.0 ng/ml/h; 48°C, D1; PVP = 7.5 pg/ml; PRA = 18.0 ng/ml/h; 48°C, D8; PVP = 2.8 pg/ml; PRA = 16.8 ng/ml/h
FINBERG ET AL.; J. APPL. PHYSIOL. 42, 654, 1977	HUMAN	EXERCISE, ERGOMETER, 1.2L/min	NO ACCL, 50°C PRA = 8.0 ng/ml/h, PA = 18 ng/100ml; NATURAL ACCL, 50°C PRA = 3.76 ng/ml/h, PA = 9.5 ng/100ml; D1, 50°C PRA = 22 ng/ml/h, PA = 34.0 ng/100ml; D7, 50°C PRA = 11.1 ng/ml/h, PA = 24 ng/100ml
ORENSTEIN ET AL. PEDIATR. RES. 17, 167, 983	HUMAN	EXERCISE, ERGOMETER, 20W INCRE-MENTS TO EXHAUSTION	38°C, CONTROL, PRE-EX, PRA = 7.6 ng/ml/h, PA = 18.4 ng/100ml; 38°C, CONTROL, POST-EX, PRA = 13.0 ng/ml/h, PA = 36.4 ng/100ml; 38°C, CYSTIC FIBROSIS PATIENTS, PRE-EX, PRA = 5.6 ng/ml/h, PA = 16.4 ng/100ml; 38°C, CYSTIC FIBROSIS PATIENTS, POST-EX, PRA = 16.4 ng/ml/h, PA = 39.7 ng/100ml
DAVIES ET AL.; J.APPL.PHYSIOL. 50, 605, 1981	HUMAN	EXERCISE, ERGOMETER, 75W	45°C, PRE-ACCL, 45MIN EX, PRA = 5.6 ng/ml/h, PA = 500 pg/ml; 45°C, POST-ACCL, 45MIN EX PRA = 7.2 ng/ml/h, PA = 670 pg/ml; 45°C, PRE-ACCL, 45MIN EX, SALT ADMINISTERED DURING ACCL PRA = 7.0 ng/ml/h, PA = 340 pg/ml; 45°C, POST ACCL, 45 MIN EX, SALT ADMINISTERED DURING ACCL PRA = 7.0 ng/ml/h, PA = 420 pg/ml
FINBERG ET AL. J.APPL.PHYSIOL. 36, 519, 1974	HUMAN	EXERCISE, TREADMILL, 4.7 km/h	50°C, SUMMER, POST-EX, PRA = 6.1 ng/ml/h; 50°C, WINTER, POST-EX, PRA = 9.0 ng/ml/h; 50°C, POST-EX, WATER AD LIB, PRA = 9.3 ng/ml/h; 50°C, POST-EX, NACL, PRA = 5.8 ng/ml/h

Reference	Species	Exercise	Results
FRANCESCONI ET AL. J. APPL. PHYSIOL. 55, 1790, 1983	HUMAN	EXERCISE, TREADMILL, 4.8km/h	35°C, 79% RH, PRE-EX, CONTROL PRA = 3.5 ng/ml/h, PA = 8.0 ng/100 ml; 35°C, 79% RH, PRE-EX, 5% HYPO, PRA = 8 ng/ml/h, PA = 13.5 ng/100ml; 35°C, 79% RH, POST-EX, CONTROL PRA = 8.5 ng/ml/h, PA = 6.3 ng/100ml; 35°C, 79% RH, POST-EX 5% HYPO, PRA = 11.8 ng/ml/h, PA = 13.5 ng/100ml; ACCL REDUCED PRA RESPONSES
FRANCESCONI ET AL. J.APPL.PHYSIOL. 59, 1855, 1985	HUMAN	EXERCISE, TREADMILL, 4.8km/h ACCL	49°C, PRE-EX, HYPO: 0% PRA = 3.0 ng/ml/h, PA = 10 ng/100ml; 3% PRA = 6.1 ng/ml/h, PA = 12 ng/100ml; 5% PRA = 7.5 ng/ml/h, PA = 16.5 ng/100ml; 7% PRA = 7.7 ng/ml/h, PA = 16 ng/100ml; 49°C, POST-EX, HYPO: 0% PRA = 5.2 ng/ml/h, PA = 12.2 ng/100ml; 3% PRA = 7.4 ng/ml/h, PA = 18.2 ng/100ml; 5% PRA = 9.2 ng/ml/h, PA = 23.5 ng/100ml; 7% PRA = 9.2 ng/ml/h, PA = 20.0 ng/100ml;
KIRSCH, ET AL. EUR. J.APPL.PHYSIOL. 47, 191, 1981	HUMAN	ENDURANCE, 27-32KM	PRE-EX, PRA = 0.17 ng/ml/h; POST-EX, PRA = 0.6 ng/ml/h; 45MIN POST-EX, PRA = 0.33 ng/ml/h
FRANCESCONI ET AL. EUR.J.APPL.PHYSIOL. 51, 121, 1983	HUMAN	EXERCISE, TREADMILL, 5.6km/h	CONTROL, PRE-EX PA=16.5 ng/100ml; 45°C, POST EX PA = 23 ng/100ml; 45°C, POST EX, ALBUM INFUSION PA = 16 ng/100ml

It is noteworthy that in most of these studies addressing intercompartmental fluid shifts in acclimated, non-acclimated, heat tolerant and heat intolerant test subjects, potential endocrinological effectors have not been investigated. In fact, while a wide variety of physiological, pharmacological, and physical factors have been identified as predisposing an individual to heat/exercise injury (155), we are unaware of any studies which have evaluated the association between these factors and endocrinological adaptations to exercise in the heat.

For example, we have recently compared the physiological and hematological responses of older and younger men during 10 d of heat acclimation (124). While endocrinological responses are still being evaluated, the results of this study indicated no impairment of thermoregulatory responses when test subjects were separated by 25 years in age, but matched for VO_2 max and selected morphological factors. Alternatively, when Paolone et al. (125) compared the responses of young (26 y) and older (58 y) men to exercise in the heat, they observed that PRA was generally more markedly increased in the younger group. They further concluded (125) that increased PRA in the younger population may have contributed to their increased ability to protect PV during the first 60 min of exercise in the heat.

Additional studies on the role of endocrinological responses in the fluid shifts of heat acclimation, heat intolerance, and factors which predispose to heat injury may be useful in the design of pharmacological interventions to reduce the physiological strain of exercise in the heat. For example, although atrial hormone has been shown to counteract the vasoconstrictive effects of PRA and the anti-natriuretic effects of PA (111), to our knowledge no studies have been conducted assessing its response during heat exposure or exercise in the heat. We have demonstrated in a small animal model that during moderate (10%) hypohydration PV is protected by an influx of water from the interstitial fluid compartment, and at 15% hypohydration, both the interstitial and the intracellular compartment contribute to the maintenance of PV (42). However, no studies of hormonal control, if existent, of these intercompartmental shifts have been reported. In a much earlier study Giec (72) estimated the source of the water content of sweat during 2 h of sedentary heat (50°C) exposure and concluded that 66% originated in the interstitial fluid, 18% in the PV, and 16% in the erythrocytes and other cells.

Yet another understudied area is the complex series of endocrinological responses occurring in females during exercise in the heat with respect to circadian periodicity and phase of the menstrual cycle (88,158); for example, Horvath and Drinkwater (92) have reported a greater decrement in PV following exercise in the heat during the luteal phase of the cycle. Grucza et al. (78) have observed several significant differences in sweating responses to heat exposure between men and women yet to our knowledge no studies have been executed to investigate the endocrine correlates, if extant, of these differences with respect to either the monthly cycle or aging.

CONCLUSION

In this review we have attempted to address comprehensively the hormonal adaptations and responses which function in the maintenance of fluid and electrolyte homeostasis during heat/exercise stress. Inconsistencies are apparent, but even more obvious are the myriad of experimental parameters which can affect experimental results. The exercise or, indeed, the heat may range from mild to severe, the time of exposure from acute to chronic, the fitness status of the subjects from poor to elite - clearly, all these can affect qualitatively and quantitatively adaptational profiles and response

patterns. Add to these the variables that can be more subtle or difficult to assess - hydration status, acclimation level, prior dietary history, and it is not difficult to envision persistent inconsistencies or even apparent contradictions. However, frequently, a close reading of the methodology will reveal logical and valid reasons for what at first glance appear to be contradictory reports. In fact, in reviewing the literature over the past 40 years, one of the several striking differences to be noted is the more comprehensive and complete descriptions of the test scenarios, experimental conditions, test subjects, and methodologies that are currently provided. It is incumbent upon all investigators to maintain and expand this careful attention to experimental and methodological details so that data can be properly evaluated.

It would have been useful, if it were possible, to correlate plasma hormonal response patterns and intensities to fundamental physiological variables relevant to exercise/heat stress. Information on indices related to physiological strain (e.g. rectal temperature, heart rate, osmolality, sodium levels, plasma volume as estimated from hematocrit-hemoglobin changes) may have permitted comparisons of the endocrine response patterns with these physiological variables across experiments. For example, the endocrinological responses to sedentary (e.g. 1 h, $60^{o}C$, euhydrated) heat exposure or exercise (e.g. 15 min $30^{o}C$, 3% hypohydrated, 50% VO_2max) in the heat provide useful, but independent, information on human adaptations to exercise/heat stress. If in these two experiments, however, the response patterns could be related to common physiological criteria as indicated above, then much more comprehensive and useful interpretations could be made. For example, the intensity of the hormonal responses could be related to the physiological criteria affecting this intensity in both experiments. Conclusions might be drawn as to hormonal responses which contribute to the successful completion of either exercise or the heat stress in both experiments, and the role of baroreceptors, osmoreceptors, volume receptors and thermoreceptors could be assessed. The degree of stress would be related to a physiological rather then an environmental/exercise criterion, and the diversity of environmental/exercise criteria could be more comparable.

In closing, we would like to reemphasize that despite progress in understanding the role of hormonal action in regulating fluid and electrolyte balance during heat exposure/exercise in the heat, much remains unknown. A recent brief review by McDougall (114) underscores this. In this succinct report McDougall (114) discusses the factors which are instrumental to the control of aldosterone secretion. He notes that this hormone is secreted only by cells of the zona glomerulosa of the adrenal cortex. Further, he reports that angiotensin II, adrenocorticotropic hormone, potassium loading, and sodium deprivation are all stimulatory to aldosterone secretion while atrial natriuretic peptide is inhibitory. Despite these well-established regulatory factors, McDougall (114) asserts that several hypotheses and observations regarding the control of aldosterone biosynthesis remain unconfirmed and sometimes confusing: the role of monoamines and other hormones, alterations in adrenal sensitivity, occasional dissociation of the responses of angiotensin II and aldosterone, central stimulation of aldosterone secretion. McDougall concludes that much further in vitro and in vivo work is necessary "for full understanding of the physiological control of aldosterone secretion". More generally, we also conclude that much further work is necessary for a complete understanding of the role of the endocrine system in maintaining fluid and electrolyte homeostasis during the challenge of heat exposure/exercise in the heat.

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